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THE DIAGNOSIS AND TREATMENT OF CARDIOVASCULAR-RENAL DISEASE.[1]

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The meaning of the term cardiovascular-renal disease is evident. That such an association is of frequent occurrence every practitioner of experience knows. There are, however, differences in the morbid states and in the mode of invasion which influence both prognosis and treatment, and which are still imperfectly understood by many. I have for many years taken opportunity to emphasize these differences, but as I am constantly reminded in my relations with physicians that efforts in this direction have been only partially successful, I have decided to ask the indulgence of this influential body while I consider the subject somewhat further.

To begin with the simplest and best understood of the processes, it was noted almost as early as Bright recognized the varieties of morbid states associated with his name, that in a certain number of them there was hypertrophy of the heart with thickening and hardening of the arteries. Bright himself refers to both. To the arterial condition the name of arteriosclerosis was later applied and continues to be in general use. As in the further evolution of our knowledge it has been found to occur in two forms, a diffuse and a localized form, I may add that in using the term I intend the more diffuse variety, that to which von Basch has applied the name angiosclerosis, invading the vascular system as a whole and especially the smaller vessels. To the present day an explanation of this process satisfactory to all has not been vouchsafed. Bright ascribed the cardiac hypertrophy to overaction of the heart exerted in response to a direct cardiac irritant, or to overcome a resistance in the capillaries. This stimulus and resistance he ascribed to an altered quality of the blood consisting in the accumulation of toxic substances arising from defective renal excretion. Sir George Johnson elaborated the idea of a hypertrophic process in the muscular coat and transferred the irritation to the smaller arteries which further reacted upon increased effort of the heart to overcome the resistance. So far as I can learn from an examination of Bright's[2] original papers, his reference seems to be wholly to the coarser atheromatous changes which occur in the larger blood-vessels, including the aorta, and in other diseases as well—changes which are visible to the naked eye. Bright's views on the vascular changes may therefore be disregarded. In 1877 Ewald suggested an interesting modification of Johnson's theory. He also found a hypertrophy of the muscular coat of the systemic arterioles which he ascribed to increased viscosity of the blood and resulting friction in the capillaries.[3] The cardiac hypertrophy he ascribed as did Johnson to the cardiac effort to overcome the resistance in the arterioles. Though of recent authors, Johnson's pupil, Nestor Tirard, still holds to his master's view, it has been generally discarded for the more modern one, that the changes are primarily irritative, beginning at times in the media or muscular coat and extending thence to the intima and external coats, at others in the intima, always first in the arteries of small size.

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[1] Read at a meeting of the Kings County (New York) Medical Society, April 16, 1907.

One would suppose that the changes should always begin in the intima which is next the irritant blood, but that they begin also in the media is established and confirmed by recent experimental studies of R. A. Pearce and E. Mac D. Stanton[4]. It has been alleged, too, that the lesion in the media is essentially the result of injury to the elastica, the changes in the intima constituting a repair process whose object is to compensate for the weakened media and the widened lumen. It may be through the vasa vasorum that the middle coat is thus attacked by the toxic constituents of the blood. Or the thickening of the media may be the result of over pressure due to spasm excited by irritating toxins.

Confirmatory evidence of the irritant nature of the cause is found in the fact that the thickening and hardening increase with the duration of the renal disease, not always, however, in a corresponding proportion. Moreover, other conditions associated with toxic states of the blood are attended by like changes in the arteries. Such are diabetes mellitus, syphilis, alcohol, lead poisoning, the poison of gout and other products of overfeeding and overdrinking, overexertion, and especially overexertion with an excessive use of alcohol, as illustrated in workmen and drivers employed in breweries where enormous quantities of beer are drunk. To the hyperplasia succeeds in various degrees degeneration, usually fatty but sometimes calcareous, resulting in weakness of the vessel walls and occasional rupture, especially in the bloodvessels of the brain. This process extends also to the bloodvessels of the kidney in contracted kidney, but not as a rule in chronic parenchymatous nephritis.

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[2] Bright's Medical Reports, 1827, and his later paper in vol. i of Guy's Hospital Reports, 1836, Cases and Observations Illustrative of Renal Disease, etc.

[3] Virchow's Archiv, vol. lxxi.

[4] Experimental Arteriosclerosis, Transactions of the Association of American Physicians, vol. xx, 1905.

Almost invariably associated with the sclerosis of the vessels is hypertrophy of the left ventricle induced by the effort to overcome the arterial resistance and probably also as already mentioned to the direct stimulus of an irritating blood. This resistance is a complex factor, including first that of the narrowed and non-elastic vessel wall; second, the increased tension causing increased intravascular pressure and possibly also a compensatory action assumed by the heart in order to increase the secretion of urine by forcing more blood through the kidney. Nervous strain such as characterizes the strenous life of the modern business man has come to be regarded as a cause of high tension, itself a cause of sclerosis. My colleague, Dr. Alfred Stengel, who has written two or three good papers on this subject would even include neurasthenia and shock among the nervous causes operating in this way. The toxins of the acute infectious diseases may have an effect, while heredity plays an important role. In a noteworthy paper Dr. O. T. Osborne[5] suggests that certain internal secretions especially those of the thyroid gland and suprarenal capsules may be responsible for a high tension which will cause arteriosclerosis. Since the thyroid contributes a vasodilator substance and the suprarenals a vasoconstrictor stuff, an increase of tension may be caused by a diminution of thyroid secretion and an excess of suprarenal. Josué and Gouget, and after them Pearce, produced arterioscleroiss by the intravenous injection of adrenalin in rabbits. In view of the recently discovered relation of the suprarenal secretion to glycosuria we may have a further explanation of the high degree of arteriosclerosis so frequent in diabetes mellitus. The cardiac hypertrophy affects both the left and the right ventricle, but more frequently the left, as shown by the following from a recent paper by Dr. Nathaniel Bowditch Potter.[6]

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[5] The Etiology and Treatment of Arteriosclerosis, New York Med. Jour., August 20, 1904.

[6] Cardiac Hypertrophy as Observed in Chronic Nephritis, Jour. Amer. Med., Assoc., October 27, 1906, vol. xlvii.

286 *autopsies*, 151 *cases of nephritis.*

	Per cent. of hyp. of L. V.	Per cent. of hyp. of L. and R. V.	Per cent. of normal or Atro-heart.
66 chronic interstitial	30.5	11.5	58.0
22 arteriosclerotic interstitial	27.0	9.0	64.0
25 chronic parenchymatous	12.0	4.0	84.0

Recent observations go to show a larger number of cases in which all the chambers of the heart were hypertrophied.

While arterial sclerosis, recognized by a sharply accentuated aortic second sound, hypertrophy of the left ventricle with the apex displaced to the left, hard sclerosed radials, increased tension and increased blood pressure may arise quite independently of renal disease, I still hold with others that it may be also the direct result of such disease, and I may say any form of renal disease sufficiently prolonged; it is much more frequently associated with the chronically contracted kidney. Cardiac murmurs are conspicuously absent. Indeed, the presence of such murmurs removes the diagnostic value of the hypertrophy, since they point to other causes of it. On the other hand, the first sound is strong and prolonged until toward the end, when it loses its muscular character and becomes shorter and sharper. The few instances of chronic renal disease in which hypertrophy does not occur are those in which there is debility with defective nutrition, when the heart is too weak to make the extra effort required to produce hypertrophy. These cases are generally found in old persons and others whose vitality is low. Sir William Gull,[7] in a clinical lecture on Chronic Bright's Disease with Contracted Kidney, at Guy's Hospital, in 1872, referred to this form in the following language: "And here it should be observed that the kidney may undergo fibroid contraction and disorganization, and cause death by uremia, without being associated with change in the heart and arteries upon which I desire to discourse in this lecture." He continues: "In such cases the renal disease is probably due to some hereditary or congenital defect in the kidneys themselves, and is more strictly a local affection; and although the results, so far as the kidneys are concerned, may be very much like, or even apparently identical with, the more morbid conditions arising under other circumstances, what constitutional accompaniments there are, are widely different from such as occur in the disease now spoken of," referring to the condition of general arteriosclerosis (arteriocapillary fibrosis), under consideration.

Reference has been made to other causes than renal disease responsible for the arterial thickening and secondarily cardiac hypertrophy. Indeed, there are those who claim that renal disease is never the cause of arterial sclerosis, but that, on the other hand, it and especially renal contraction, with its fibroid overgrowth, is the direct result of such sclerosis of the bloodvessels of the kidney shared with the system at large, and that the cardiac hypertrophy is the result of resistance as before. To prove this was the object of the classic papers of Gull and Sutton on "Chronic Bright's Disease with Contracted Kidney (Arteriocapillary Fibrosis), published in 1872.[5] This may be the case with the arterial sclerosis and the associated renal contraction of old persons, and I repeat that I am ready to admit a primary general arteriosclerosis like that contended for by Gull and Sutton, but I still hold that many cases of arterial sclerosis are secondary to a primary renal contraction, although perhaps not so often as once supposed. The result is, however, the same, combined vascular and renal disease in which the cardiac hypertrophy is secondary to the arterial sclerosis.

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[7] British Med. Jour., December 21, 1872. See also collected writings.

If I am asked to point out clinical differences between these two varieties of sclerosis I find it difficult. Both are equally insidious, in both there is the absence of dropsy except in the very last stages of the form with primary renal sclerosis, though here it is also rare while it is wanting throughout in general arteriosclerosis. In both there is a scanty albuminuria with very few hyaline casts, but if the opportunity presents to study the urine for a time I should say the albumin and casts appear earlier in the primary renal cases. Indeed, in general arteriosclerosis there is often no albuminuria whatever, although hyaline casts are present. In general arteriosclerosis there are often brain symptoms due to anemia and imperfect circulation, namely, vertigo, tinnitus, and amaurosis. In addition to this there is often met a set of symptoms simulating those of uremia, but still not uremia. In the latter condition retinal changes occur early, but they are not those of retinitis albuminurica and hemorrhage into the retina. As pointed out by de Schweinitz there is thickening of the arteries which compress the veins where they cross them. These changes may occur early. Retinitis albuminurica, hemorrhages, and more serious derangements of vision occur only when the kidney lesion is primary. An important symptom characteristic of general arteriosclerosis is a tendency to emaciation and loss of weight, a change of color, a pallor, and loss of vigor which is characteristic. The condition of the arteries interferes with an adequate nutrition and the patient wastes as well as grows weaker and anemic. Dr. Alfred Stengel suggests that this anemia is apparent rather than real, since the blood in the vessels is simply obscured by the thickened vessel walls.

Perhaps the most characteristic difference is found in the blood pressure. In general arteriosclerosis blood pressure may be increased, but not nearly as much as in renal sclerosis when the systolic pressure is often 170 to 220 mm. as contrasted with an average normal of 145 mm.

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[5] Medico-Chirurgical Transactions, 1872, vol. iv, p. 273. Reprinted in a collection of the writings of Sir William Gull, New Sydenham Society, 1904.

An important etiologico-pathological difference is claimed by Gull and Sutton in the kidneys. According to them the destruction of renal tubules is due to the contraction of new connective tissue, the formation of which is due to the arterial changes. In primary contracted kidney the renal parenchyma dies first and is replaced by the connective tissue secondarily.

These instances illustrate two categories of associated renal and cardiovascular disease: (1) That in which the kidney disease is primary; and (2) that in which the renal condition is a part of a general arteriosclerosis. In both classes the cardiac hypertrophy is secondary.

On the other hand kidney disease may be the direct result of heart disease. This starts in and continues largely in a passive congestion caused by the backing of the venous blood into the kidney. It occurs in mitral valve disease, both stenosis and incompetency, myocarditis without valvular lesion, and sometimes also in acute dilatation. The result of the first two is the kidney of chronic passive congestion or cyanotic induration, a well recognized condition, in which there is eventually some overgrowth of interstitial connective tissue with resulting hardening; while the third, acute dilatation, causes an acute passive hyperemia of the kidney. The symptoms of passive congestion when present in mild degree are a scanty dark hued urine of high specific gravity, containing a little more than a trace of albumin, and either no casts at all or a few hyaline casts, more rarely moderately granular. In severe degrees the albumin is much increased, while the casts commonly remain the same kind though increased in number. A few red blood disks may now also be found by the microscope, but there is never enough blood to be visible to the naked eye. It is these cases which are still too frequently regarded as cases of primary kidney disease by the hasty observer, and it must be admitted that the diagnosis is not always easy, at least at the first examination. There is dropsy with albuminuria and casts, three symptoms deemed by many physicians all that is necessary to justify the diagnosis of Bright's disease. The cases which are most difficult to separate from Bright's disease are cases of myocardial disease with failing heart without signs of valvular lesion in which stasis has increased until dropsy and albuminuria have become marked. In these cases of primary heart disease arterial sclerosis is much more rare. The disease is cardio-renal in the strict sense of the term.

The forms of Bright's disease which are most frequently confused are chronic parenchymatous nephritis and such cases of chronically contracted kidney in which hypertrophy has become dilatation and the accentuation of the aortic second sound has grown feeble, while dropsy, a rare symptom in the earlier stages of contracted kidney, has supervened.

The first essential to a correct diagnosis is, therefore, a deliberate examination. The exact condition of the heart must be ascertained in each case, and if a mitral murmur is present, if the urine is scanty, of high specific gravity, even if it contain tube casts, the chances are almost all that it is a primary heart disease. If there is no murmur the difficulty is greater. But even in the absence of a murmur if the clinician will but wait a little while before making a diagnosis he will avoid error. Especially valuable in these cases is the scanty, dark colored urine of high specific gravity. Marked shortness of breath is invariable in any form of heart disease and occurs early, whereas in primary renal disease it is more frequent and occurs later. Most valuable is the information given by the heart sounds. In primary renal disease there is sharp accentuation of the aortic second sound, while in primary heart disease this is wanting, and there is accentuation of the pulmonic second sound. The blood-vessels must be carefully examined and the presence or absence of sclerosis determined, bearing in mind that this condition is much more likely to be present in primary renal disease than in primary heart disease. At the present day no examination is complete without estimating the blood pressure. In primary renal cases of either kind it is commonly high, but higher in primary renal sclerosis; in mitral valvular disease it is low, at least the disatolic pressure is low even though the systolic be high.. Fortunate are we if the history of the case from an early stage is available. Too often it is wanting, especially in hospital cases.

A diagnosis is of extreme importance because prognosis is widely different and treatment by no means identical. The prognosis is much more favorable when the heart disease is primary, even when there is extreme dropsy, and I have seen patients improve incredibly even when they have been almost moribund. In the cases referred to of primary contracted kidney with hypertrophy, in which the heart muscle has given way and the hypertrophy has been replaced by dilatation, resemblance is close, but by this time the heart is very much larger than in primary heart disease, in which, too, the enlargement is also to the right, while in primary renal disease the hypertrophy is to the left only. In this condition, too, there remains almost to the very end a certain degree of accentuation of the aortic second sound and the blood pressure continues high, much higher than in mitral valve disease. We may be aided by uremic symptoms. Uremic symptoms are much rarer in the cases beginning as heart disease than in those beginning as renal disease or general arterial sclerosis, indeed very rare. Such symptoms point, therefore, to the latter condition. But I often find that the practitioner interprets as uremic, symptoms otherwise explainable, and sometimes it is not easy to say whether they are uremic or not.

Now as to treatment. To begin with the cases which may be classified as more easy of diagnosis, in which there are signs of chronic interstitial nephritis, commonly polyuria, small albuminuria, no dropsy, hyaline and pale granular casts not very numerous, hypertrophy of the left ventricle, sclerosed arteries, and high blood pressure, 160 or more mm. of mercury. Are there curative measures? I fear not curative in the strict sense of the term but certainly helpful. Whether the process has started in the bloodvessels as a whole or began in an interstitial inflammation of the kidney, toxic substances have been at work irritating the renal tubules and the intima of the arterioles, impairing the integrity of both. Such substances have arisen from over consumption of any kind of food, but especially proteid foods and alcoholic drinks, perhaps also tea and coffee. These, therefore, must be cut out of the dietary and substituted by milk and cereals and vegetables. If there be constitutional syphilis, it must be treated; if diabetes, the sugar removed from the blood; if gout, the uric acid and purin bodies eliminated. The last is accomplished by excluding proteid substances, including especially glandular organs such as sweetbread, liver, and kidney, which are especially rich in purin bodies. If the patient is overworked mentally or physically he must rest.

Can we remove the sclerosed vessels, overgrown connective tissue in the kidney, and the hypertrophied heart by drugs? I fear not. But we may counteract their effect and perhaps retard their increase. The iodides not only have the tendency to remove overgrown connective tissue but also to act as permanent vasodilators. Vasodilatation is especially desirable. How far it is possible I do not know; indeed, it has lately been denied, but I am in the habit of using the iodides with seemingly good effect. They may act, too, as is claimed by some by diminishing the viscosity of the blood, which may be one of the causes of cardiac hypertrophy. I prefer the iodide of sodium given in doses rarely large but commonly as large as can be borne without disadvantage. My doses never exceed 10 grains three times a day. More frequently they are less, but they must be kept up continuously. When there are retinal symptoms (retinitis albuminurica) I substitute the biniodide of mercury in $\frac{1}{48}$ to $\frac{1}{24}$ grain doses. In spite of this the conditions slowly grow worse, although I think they may be retarded.

As to other treatment, it must consist mainly in combating symptoms. Digitalis is not, as a rule, indicated; indeed it may be harmful. Should the cardiac action become too powerful tincture of aconite in three to five drop doses, three times a day or oftener, will calm the unruly organ, especially if associated with rest and a reduced amount of food. Nitroglycerin, reputed a prompt vasodilator, may be used under the same circumstances in doses of $\frac{1}{100}$ to $\frac{1}{50}$ grain at short intervals, as often as once in two hours, and is sometimes advantageously kept up for a long time at longer intervals. Nitrite of sodium may be given in 3 to 5 grain doses as often. I do not think the nitrites are as satisfactory as aconite when there is powerful cardiac action. On the other hand, they sometimes produce an aggravation of the symptoms, producing an unpleasant fulness in the head, to which however, the patient soon becomes accustomed.

General massage is a means of treatment which is often beneficial, helping the circulation and easing the work of the heart.

However unsatisfactory the treatment for this class of cases may be, it can be for the most part intelligently directed because the diagnosis, as a rule, has been correctly made. It is not so with the other divisions, namely, those associated with dropsy and large albuminuria and casts, which may be due to valvular disease or myocardial disease, to parenchymatous nephritis or the last stages of chronically contracted kidney. Fortunately the treatment is not widely different; indeed, up to a certain point it is identical. Thus, whichever of the conditions named is present, the patient must be put at rest in bed. This is sometimes difficult to carry out. He has perhaps been sitting up night and day, and imagines he will suffocate if he goes to bed. The bed should, however, be insisted upon, and in twenty-four or forty-eight hours he will be convinced by the results. He should also be put on a milk diet. Here, however, the treatment of the two classes of cases begins to diverge. If the case is one of primary heart disease, liquids should be restricted, although they may still be the best form of food. The patient's tissues are water logged, and we want to reduce the water rather than add to it. Yet I have seen such patients deluged with liquids, because, in the opinion of the physician, it was necessary to pass water through the patient to wash him out and thus diminish the danger of uremia. But this particular patient is not in danger of uremia, and to sluice him with water tends to aggravate a symptom we seek to abate. The liquid ingesta must be cut down. Nay, more liquids must be removed from him. He must be purged with hydragogue cathartics, the best way to get fluid from him. He must be sweated by hot-air or vapor baths or by hot packs, and the secretion of urine stimulated by diuretics if possible.

Here, too, we may expect diuretics of a class to act which in advanced renal disease are useless, viz., diuretics of the theobromin class which act directly on the renal epithelium and require therefore a certain integrity of this tissue for the best results. Of these, besides theobromin and caffein, there are three—diuretin, theocin, and agurin. Theobromin is a drug difficult to dissolve, but, nevertheless, I have seen it produce free diuresis in cardiac cases—given in simple admixture with water in doses of 45 grains in twenty-four hours. Pharmaceutical chemists have therefore sought to produce soluble compounds of this substance. Such are diuretin, agurin, and theocin. Caffein is a good representative of this class and has the advantage of being a cardiac and cerebral stimulant also. It is a soluble derivative of the xanthin bodies, a trimethylxanthin, while theobromin is a dimethylxanthin and is insoluble. I have used caffein with great advantage in conjunction with digitalis and its congeners which act by increasing blood pressure and which are suitable for both renal and cardiac disease. A favorite treatment with me in such cases is to alternate digitalis and caffein, especially caffein sodio-benzoate, giving each every four hours, alternately, suiting the dose of each to the case; say of tincture of digitalis 5 to 10 m nims, and of caffein citrate 3 to 5 grains. The citrate of caffein must be given in larger doses than caffein, one-third larger. Caffein sodio-benzoate is suited for hypodermic use. Of late I have been substituting the fluid extract of digitalis for the tincture.

Theobromin has the advantage of being more prolonged in its action than caffein, but it is not so well borne by the stomach. The same is true of diuretin, a soluble combination of theobromin (50 per cent.) with salicylate of sodium. Diuretin is also an uncertain drug. Two or three years ago I received from a medical friend in Boston a useful hint on the use of diuretin. He informed me that as found in the shops it is often inefficient because spoiled, in which event it makes a turbid solution. The drug should therefore be thus tried, and if it does not dissolve in clear solution it should be discarded. On the whole I have been rather disappointed in diuretin, whose effect is moreover temporary and in my experience taxes the stomach severely. It is given in doses of 5 to 15 grains three to four times daily.

A far better derivative of theobromin is theocin or theophyllin or dimethylxanthin sodium. Theocin is an alkaloid which exists in small amount in tea leaves, discovered by Kossel in 1888. The quantity is, however, so small as to be unavailable. It has therefore been produced synthetically by Bayer, who also makes a soluble double salt of acetate of sodium and dimethylxanthin, acet-theocin sodium or soluble theocin, a preparation more efficient and less irritating to the stomach. It is given in doses of 4 to 8 grains three times daily for an adult, in solution or tablet form, or half as much twice as often. When successful prompt diuresis results, the urine increasing to 70, 80, 100, or more ounces daily. As compared with theobromin again its effect is less persistent, and with caffein it lacks the effect of cardiac stimulation. Another preparation which seems to differ only in the quantity of dimethylxanthin is agurin, with which I have as yet had no experience although it is an older remedy than theocin. All these remedies require a certain degree of integrity of the kidney and are therefore much more efficient in primary heart disease than in primary kidney disease; in fact, they are of little or no use in advanced renal disease, whence their inertness has some diagnostic value.

As to the withdrawal of liquids you may ask, How is this accomplished by a milk diet? I answer, by restriction of the amount ingested; easier done with milk than any other shape of food, while it is moreover in an easily assimilable shape. We may begin with

2 ounces every two hours, which will make but 24 ounces in the twenty-four. Such a patient is not hungry and will not cry for more. Nor is he in danger of starvation. He will feed on his serous effusion and in a few days he will begin to be hungry. I have frequently noted the beginning of happy results when the patient begins to be hungry. The serous effusion will start to disappear. Before this, however, the tension should have been removed by purgatives and sweats and the secretion of urine in increased quantity have set in. Even in cases of pure renal origin I think the matter of fluid ingestion is sometimes overdone, and I have had to insist on the restriction of liquid in these cases in order to remove dropsies and effusions.

It may be necessary even to add to these measures incision of the legs or the use of Southey's tubes in order to facilitate the extraction of liquid from the tissues. I prefer the former. The better method is to make a single incision an inch long behind each ankle rather than to make more numerous and smaller incisions throughout the leg. Enormous outflow of fluid is often thus secured, while the relief to tension favors the action of the diuretics.

Supposing the renal changes to be primary in these water-logged cases, the form producing these symptoms is most frequently chronic parenchymatous nephritis. The difference is greater in prognosis than in treatment, the former being much more grave. The success in the treatment of the variety just described depends almost entirely on the degree of integrity of the kidney which remains. The rest and diet are the same. The measures to relieve tension and remove the anasarca are the same as to purgatives and sudorifics, but the class of remedies so useful in producing diuresis and in stimulating renal action is comparatively useless, and the drugs which increase cardiac contraction and favor osmosis are most useful, that is as representing the former, digitalis and strophanthus, given in full doses and the same doses for each, 10 to 15 minims. It should be remembered that the infusion of digitalis is often in these cases more efficient than the tincture. Broom is another diuretic which sometimes acts very well. In times past broom tea was found efficient, but fell into disuse because of its bulkiness and disagreeable taste. It has been replaced by its active principle, spartein, in the shape of the sulphate. Many years ago I began to use spartein in the dropsy of Bright's disease and thought I was giving quite large doses when prescribing ¼ grain with such frequency as to secure two grains in the twenty-four hours. They were certainly much larger doses than heretofore griven. Now I advise it in even larger doses, ¾ grain every three hours. I have never known any harmful result and it is well borne by the stomach.

As representing the salines or osmosis promoters, the citrates, tartrates, and acetates are indicated and may sometimes be advantageously combined with cardiac stimulants in the same prescription. Purgatives and sweats are also necessary in these cases. Purgatives are especially useful also from the standpoint of eliminators of toxic solids, which the kidneys are helpless to remove, as well as water, von Noorden's experiments having shown that urinary toxins are very much more freely removed by the bowel than by the skin, and thus uremia is averted. Of aperients, every one knows that the hydragogues are indicated, including especially salines, compound jalap powder, and elaterium.

In this connection I will allude to a drug which I think merits more frequent trial, a purgative and diuretic so striking in its effect at times as to have received the name of internal trocar, and that is, apocynum cannabinum, the root of the Canadian hemp. Only recently a friend informed me that in a given case he found it so active as to cause the patient to pass a bucketful of urine in a given night. Unfortunately it is sometimes so irritating as to produce vomiting as well diuresis and purging. An active principle known as "apocynin" is less irritating. It is put up in $\frac{1}{6}$ grain granules, of which 3 are given every two hours until the effect is secured.

We must not forget an old but still excellent diuretic and eliminator sometimes producing magical effects, namely, the pill of calomel, squills, and digitalis, in doses of calomel, $\frac{1}{2}$ grain; of squills, 1 grain; and of digitalis, 1 grain, three or four times a day. I have known a dropsy which has resisted other measures to disappear with rapidity. The purgative and diuretic effect are commonly both secured. Here again it is in the anasarca of primary cardiac cases that we have the best results, although they are sometimes obtained in renal cases also.

These measures will cure a number of milder cases and once in a great many times the severer will yield. When this is the case there must be a considerable remnant of the kidney intact or nearly so. It used to be said that if we can keep our patient alive until the stage of contraction is reached his chances of prolonged life are improved, but I have of late years doubted whether this is true. In the contracted stage there is less secreting structure than in the parenchymatous stage. In this variety the danger of uremia is greater, but not nearly so likely to present itself as in contracted kidney.

Most important to remember is the fact that a diuretic which answers in one case often fails in another, and it is therefore necessary to have a full quiver of remedies to be drawn upon judiciously as needed.

It may be expected of me to say something of the influence of chloride retention upon œdema and albuminuria. Strauss in Germany and Widal and Javal in France, in 1902 and 1903, called attention to the fact that there is an abnormal quantity of sodium chlorid in the tissues of persons affected with dropsy, reporting cases in which it was possible to cause œdema to appear and disappear almost at will by varying the amount of chloride in the diet. The rationale of these events is supposed to be as follows: In nephritis the ability of the kidney to excrete chlorides is decreased, whence their accumulation in the tissues and the consequent accumulation of water in the same tissues in order to keep the chloride in solution. Although there appear to be exceptions and the parallelism is not always demonstrable, the truth in general of the statement seems to be admitted. An opposite theory, that the primary trouble in nephritic œdema is the retention of water to which the retention of salt is secondary, is not sustained by the weight of evidence, which goes to show that the chloride retention is primary and the water retention secondary. Chloride retention has been observed in some cases of cardiac dropsy and in some cases of œdema occurring in patients with inflammatory disorders, but there is no evidence to show that there is any causal relation between the two conditions or that the chloride retention is more than can be explained by the retention of fluid in the œdematous parts.

As to the treatment of renal dropsy growing out of these facts, it would naturally consist in the elimination from the diet of food rich in chlorides, which enables the kidney to remove with ease the remnant remaining in the tissues. It is advised to reduce the chlorides ingested to 2 or 3 grams daily. This is accomplished by a diet including eggs, unseasoned meat, unsalted butter and milk, bread without salt, fresh-water fish, potatoes, rice, fresh vegetables, fruits, and chocolate.

THE TREATMENT OF PNEUMONIA, ESPECIALLY BY OUTDOOR AIR.

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In 1906 there were treated in the Presbyterian Hospital in New York 128 cases of acute lobar pneumonia. The patients were of every age and the cases were of every sort, many of them alcoholic, and many constituting the terminal factor in chronic diseases of necessarily fatal type. An interesting fact is that among the recoveries were 47 patients (or 36.7 per cent. of all the cases) who received absolutely no medicinal treatment whatever, except the occasional laxative which any bedridden febrile patient may require. No cardiac stimulants were given, no hypnotics, no vaso-dilators, no inhalations, no empirical remedies. These patients were for the most part treated with good fresh outdoor air throughout their entire illness. They were placed either upon a roof or in small rooms where the artificial ventilators were sealed up and the heads